A STUDY OF FACTORS INFLUENCING MAJOR AMPUTATIONS IN DIABETIC FOOT

DISSERTATION SUBMITTED FOR

BRANCH – I M.S (GENERAL SURGERY)
APRIL 2013

THE TAMILNADU
DR.M.G.R.MEDICAL UNIVERSITY
CHENNAI

CERTIFICATE

This is to certify that this dissertation titled **"FACTORS INFLUENCING MAJOR AMPUTATIONS IN DIABETIC FOOT"** submitted by **DR.G.DINESH KANNAN** to the faculty of General Surgery, The TamilNadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MS degree Branch I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from January 2011 to December 2012.

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DECLARATION

I**, DR.G.DINESH KANNAN** solemnly declare that the dissertation titled **"FACTORS INFLUENCING MAJOR AMPUTATIONS IN DIABETIC FOOT"** has been prepared by me. This is submitted to **The TamilNadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MS degree (Branch I) General Surgery.

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ACKNOWLEDGEMENT

I am greatly indebted to my unit **Chief Prof.Dr.D.Maruthupandian.M.S**, and **Prof.Dr.D.Soundarajan.M.S.,** Professor and Head of the Department of General Surgery , Government Rajaji Hospital , Madurai for their excellent guidance in conducting this study.

I am also grateful to my retired unit Chief and Head of the Department of General Surgery **Prof.Dr.M.Gobinath.M.S** for his help and guidance.

I express my gratitude to my unit Assistant Professors **Dr.K.Karunakaran.M.S., Dr.R.Ganesan.M.S., Dr.D.Latha.M.S.D.A.,** for their guidance throughout this study.

I also express my gratitude to **Prof.Jeyakumar.M.S.M.Ch. ,** Head of the Department of Vascular Surgery and **Prof.Arthur Joseph Asirvatham. M.D.D.Diab**., Head of the Department of Diabetology for their guidance in going forth with this study.

I express my gratitude to **Dr.N.Mohan.M.S.,** Dean , Madurai Medical College, Madurai and **Dr.Edwin Joe .M.D.**, former Dean , Madurai Medical College, Madurai for permitting me to use the clinical materials for my study.

I extend my sincere thanks to all the patients who willingly submitted themselves for the study.

CONTENTS

INTRODUCTION

Diabetes is a common disease causing lower extremity amputation throughout the world, particularly in India, the Diabetic capital of the world[1]. By 2025, it is estimated that India will have world's majority of diabetics. Diabetes Mellitus is the most important cause of non-traumatic amputations worldwide. Amputations are perhaps the most feared complication of Diabetes.

In 2011 around 8.3% of world population had diabetes. Among these, vast majority were inhabiting in developing nations. It is projected that by the year 2030, around 10% of the human race would become diabetics. The troublesome fact is that this disease is being diagnosed more and more in the younger population of the world. Each year, over one million amputations are being performed to alleviate disease caused by diabetes. This roughly works out as one amputation in the world every 30 seconds. The predisposing factors that lead to amputation are diabetic neuropathy, bony deformities, inconspicuous trauma, and vascular diseases. In the presence of an ulcer, local sepsis and vascular insufficiency are major causes of amputation. The presentation of lesions greatly differ based on socio-economic status, quality of foot care and usage of footwear. It has been projected that one in six diabetics living in developed nations will develop ulceration before they die. This problem is even more menacing in developing nations. Diabetic foot not only affects the

individual but also their family and the community as a whole. It causes great strain on the financial and health care infra-structure of the nation. Another cause of concern is the emergence of type 2 Diabetes in children. These cases will eventually progress to develop micro and macrovascular complications including life-threatening infections at an early age. Around 25% of these cases will be non-healing and upto 28% will end up in amputation. Investing in a scientific foot care techniques and guidelines will be more cost effective in the long run[1]. Amputations alter quality of life and longevity. Amputations are associated with an increased risk of re-amputation and at an increased mortality in first decade after amputation. Early diagnosis and prompt therapy is mandatory. A team approach can reduce the number of amputations. Adequate infra-structure and facilities are essential. However, ignorance on the part of the patients and also the health care provider has made this goal hard to attain.

AIM OF STUDY

- To determine the various factors that are influencing major amputation in Diabetic foot
- To emphasize the actions that are needed to prevent the progression of the disease.

DESIGN OF THE STUDY

- Descriptive Study

PERIOD OF STUDY

- January 2011- December 2012

SELECTION OF SUBJECTS

Inclusion Criterion

- All cases of diabetic foot undergoing major amputation at the Department of General Surgery, Govt. Rajaji Hospital, Madurai during the study period were included in the study.
- Major amputation is defined as below knee (transtibial) or higher levels of amputation.

Exclusion Criterion

- Patients with Diabetes Mellitus undergoing major amputation following trauma
- Patients who had undergone prior major amputation with sepsis in the stump
- Patients not willing to participate in the study.

CONSENT

- Informed written consent

MATERIALS AND METHODS

This study was conducted in Government Rajaji Hospital, Madurai from January 2011 to December 2012 and included 81 cases both males and females, who have been amputated in surgical ward irrespective of the age group.

The diagnosis of Diabetes was made by measuring random blood sugar value on admission. The following details were collected by a questionnaire.

- Duration of Diabetes
- Previous history of minor amputation
- Whether the habit of smoking present or not
- Educational status
- Whether patient is using footwear or not
- Previous history of minor amputation present or not.

As a protocol, patients treated in the ward with diabetic ulcer foot underwent a standardized evaluation for assessment of peripheral vascular disease and peripheral neuropathy.

- Sensory neuropathy was evaluated with tuning fork and Biothesiometer .
- Nutritional assessment was done by measuring hemoglobin and serum albumin.
- The diagnosis of lower extremity arterial insufficiency judged by both clinical and non-invasive vascular studies.

    - Clinical signs include clinical non-palpability of one or more foot pulses of the affected foot.
    - Noninvasive study included an ankle-brachial pressure index (ABPI) of <0.8. Abnormal noninvasive vascular study or suggestive clinical signs make the diagnosis of lower extremity vascular insufficiency

- Wounds with frank purulence or with signs of inflammation such as warmth, erythema, lymphadenopathy, edema, loss of function and pain were used for the diagnosis of infection . Patients with infected wounds, pus culture and sensitivity was done.
- To assess the renal function, blood serum creatinine values were taken.
- To know the presence of osteomyelitis, X-ray of the local part was taken.
- To assess the level of major amputation, we stratified all the lower extremity major amputation in the following levels
    - Transtibial or below knee amputation
    - Transfemoral or above knee amputation.

DIABETES MELLITUS-HISTORY

Diabetes as a disease started in approximately 1550BC. Egyptian Papyrus records it as a disease that causes rapid weight loss and frequent urination. Greek physician Aretaeus of Cappodoc IA(81-133AD) described it as a disease in which limbs and flesh melt down meaning "a flowing through". Galen said, Diabetes disease process has an affliction to the kidneys. The term Diabetes was coined by Appllonius Memphites. The term Mellitus was coined by Thomas Willis meaning "honey sweet" in 1675. In1776 Dapson first demonstrated excretion of large amounts of sugar in urine & circulating blood in diabetic patients . The discovery of Glycogenesis in Liver is a landmark in history of Diabetes. Claude Bernard said, diabetes is caused by high glucose synthesis, in 1800's. During 18^{th} and early 19^{th} century glycosuria has been accepted as a diagnostic feature of Diabetes due to metabolic derangement. In 1869-Islets cells were discovered in pancreatic tissue by Paul Langerhans. Pancreas as pivotal in causation of the disease was elucidated by Mering & Minkowski in 1889. Discovery of Insulin and its practical application by Banting and Best 1921 is a major milestone. Roger Hirsworth discovered two types of Diabetes in 1935. In 1950, oral medications of Diabetes came into existence. In 1961,first injection of Insulin was developed by Decton-Dickson[2]. Various researches are going on for the treatment of Diabetes.

PATHOPHYSIOLOGY OF DIABETES MELLITUS

It is a metabolic disorder characterized by chronic hyperglycemia with generalised disturbance of metabolism. The type of Diabetes are

- Type 1 Diabetes Mellitus (Insulin dependent)
- Type 2 Diabetes Mellitus (non-Insulin dependent)
- Other specific types of Diabetes
- Gestational Diabetes

TYPE 1 DIABETES

Constitute 10% of cases, previously termed as juvenile onset Diabetes or Insulin dependent Diabetes.

- It involves complex interactions between environmental, hereditary and immunological entities causing damage to the Islet cells of pancreas.

There are two subtypes

- Type 1 A- immune mediated characterized by autoimmune destruction of Beta cells.
- Type 1 B – It has tendency to develop ketosis but these patients are negative for autoimmune markers.

- In the majority, immunological markers appears after triggering the event but before Diabetes become clinically evident. The immunologic markers

are GAD (glutamic acid decarboxylase), IA-2/ICA-512. Assays for auto-antibodies to GAD-65 are available commercially.

- Although other islet cell types are embryologically similar to Islet cells, they are not affected by immune mediated destruction.
- Environmental factors that influence the disease process include viruses (eg. coxsackie and rubella), bovine milk proteins etc.

TYPE 2 DIABETES

Constitute about 80% of cases. Previously called maturity – onset Diabetes, or non- Insulin dependent Diabetes of obese and non-obese type.

- Abnormality of Insulin production and peripheral resistance are at the core of type 2 Diabetes Mellitus.
- It has genetic predisposition.There is over 90% concordance among monozygotic twins. Uniparental positivity increases risk of disease. Risk of disease is nearing 40% if biparental family history is present.
- The disease is polygenic and multi-factorial.
- The charecteristics of type 2 are inadequate Insulin secretion, over production of hepatic glucose, insulin resistence. Obesity is commonly seen type 2 DM. In the early stages, glucose tolerance is retained inspite of Insulin resistance, due to ability of the islet cells to increase

Insulin secretion. As the above two phenomenon progress, impaired glucose tolerance develops leading to overt Diabetes.

- The metabolic abnormalities include
    1. Abnormal muscle and fat metabolism
    2. Impaired Insulin secretion
    3. Increased hepatic glucose and lipid production.
- The METABOLIC SYNDROME or Syndrome X is a combination of Insulin resistance, systemic hypertension, Lipid abnormalities like hyper-triglyceridemia, truncal obesity, impaired glucose tolerance or type 2 diabetes, and cardiovascular disease.

OTHER TYPES OF DIABETES

- Hereditary defect of Islet cell function
- Defective Insulin action
- Non-Endocrine pancreatic diseases
- Drugs or chemical induced eg. Steroids, Beta- blockers
- Infections
- Genetic syndromes eg. Down's syndrome, Turner's syndrome

GESTATIONAL DIABETES

About 4% of pregnant woman develop DM due to metabolic changes during pregnancy. These woman are prone to develop Diabetes in later life.

Hyperglycemia may result from

- Reduced Insulin secretion
- Decreased glucose use by the body
- Increased glucose production

Major stimulus for both the synthesis and release of Insulin is glucose. Insulin is synthesized by the Beta cells of pancreas.

RISK FACTORS FOR DIABETIC FOOT ULCERATION

Multiple factors are involved in causation of diabetic ulcer foot. The factors are

- Peripheral motor neuropathy
- Peripheral sensory neuropathy
- Autonomic neuropathy
- Vascular insufficiency
- Hyperglycemic and other metabolic derangements,
- Limited joint mobility
- Neuro-osteopathic deformities like charcot joint
- Patient disabilities
- Maladaptive patient behaviors
- Health care system failiure[3]

UNALTERABLE RISK FACTORS

- Age
- Male sex

  The male sex has 1.6 times higher risk of ulcers.

- Previous history of foot ulceration, presence of nephropathy, presence of retinopathy has increased risk of foot ulceration[4].
- Presence of limb edema hinders circulation and increases the risk of ulcer[5].
- Presence of callosity (Ref image 1) poses increased risk. Deformities like prominence of the metatarsal heads, nail deformities (Ref Image 2), clawing of the toes(Ref Image 3), corn foot (Ref image 4), Charcot prominence or hallux valgus (Ref Image 5) associated with increased risk[6].

ROLE OF SOCIO-ECONOMIC STATUS

Poor socio-economic status, assessed by literacy, earning and profession, is a strong predictor of amputation[7,8]. A lack of education means that patients are less likely to be aware of the importance of the prophylactic steps[9]. Treatment at primary and secondary health facilities is likely to be less often, the quality of care given to the low socio-economic people is more likely poor. This is known as 'inverse care law'[10].

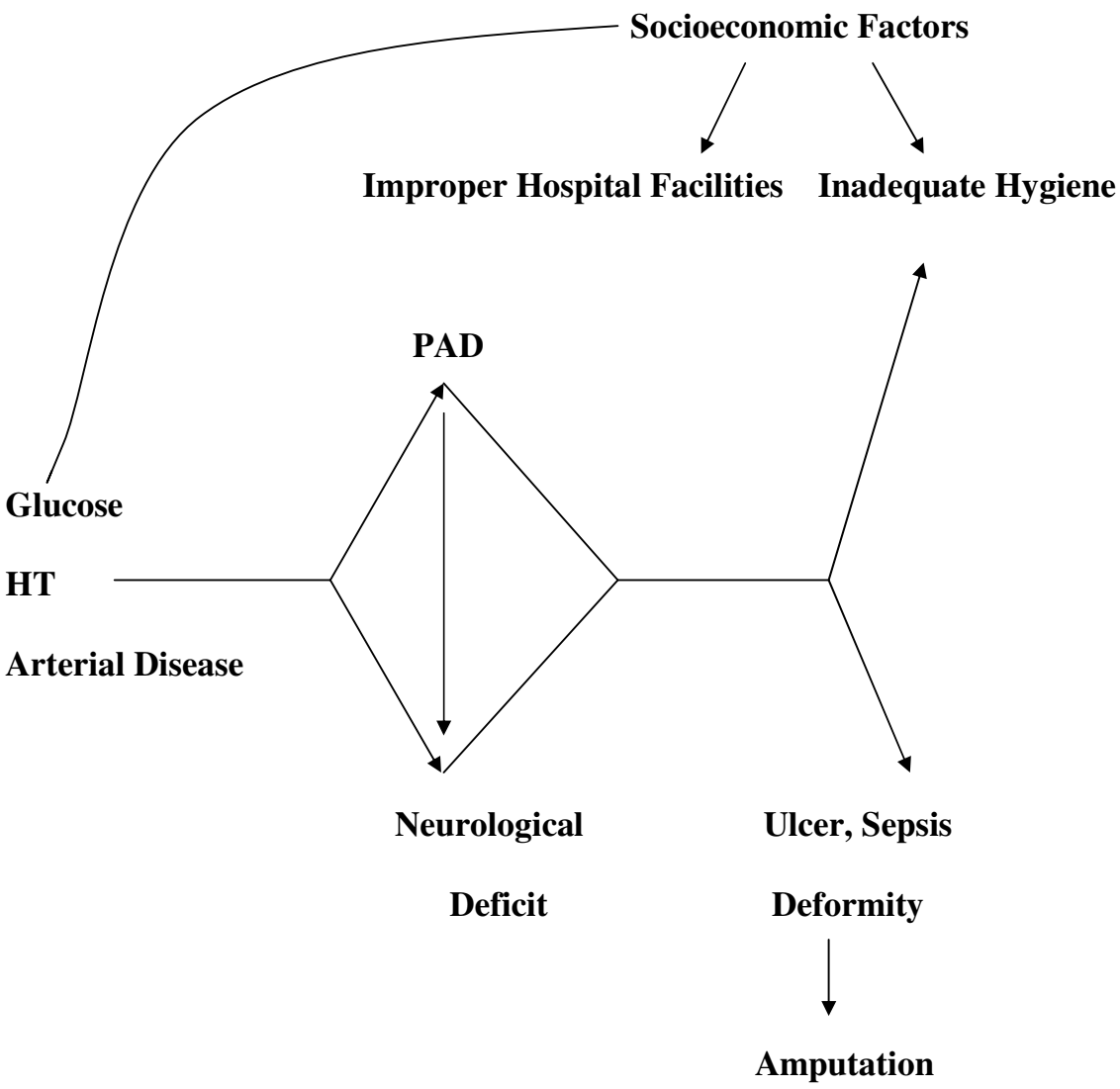
Quality health care and patient adherence to advice can normalize blood glucose levels, reduce vascular risk and normalize lipid profile.

Frequent self inspections, foot care and proper footwear reduces the chances of development of foot deformities, identifying infection or injury earlier makes treatment more fruitful.

PRECIPITATING FACTORS

- Physical trauma
- Punctured wound –thorn or nail prick
- Localized pressure from tight shoes, plastic and hard slippers
- Repeated mechanical trauma
- Heat
- Walking bare foot in hot sun

INTERACTION OF RISK FACTORS

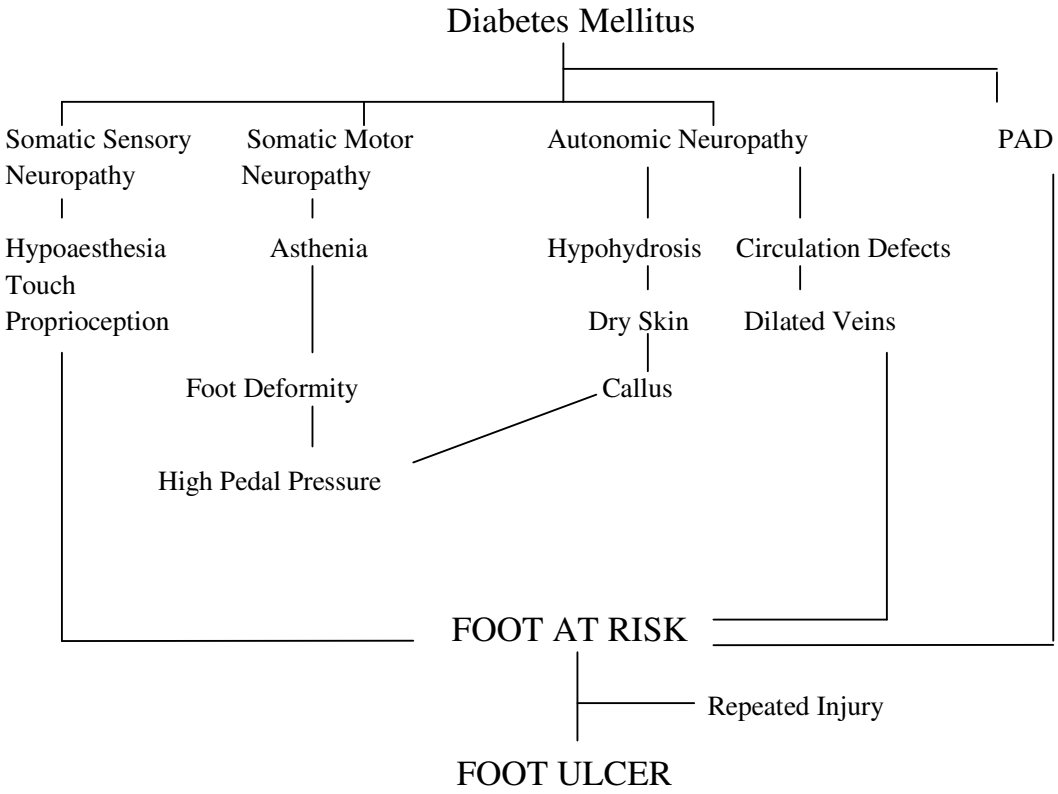


PATHOPHYSIOLOGY OF DIABETIC ULCER FOOT

The pathophysiology of diabetic foot is due to the following factors

- Neuropathy
- Angiopathy
- Mechanical stress (Ref image 6)
- Faulty wound healing
- Metabolic derangement
- Patient and provider's neglect

PATHWAY TO DIABETIC FOOT ULCERATION



DIABETIC NEUROPATHY

Polyneuropathy is one of the commonest complications of Diabetes .Most common among neuropathies in patients with diabetes are chronic sensori-motor distal symmetric polyneuropathy and the autonomic neuropathies. Sensory deficit starts in the lower limb distally and progresses to involve feet and legs in a 'stocking' pattern. This is followed by the upper extremeties in a 'glove' like pattern. Diabetes affects autonomic nervous system also. As the disease progresses wasting of the small muscles of the hand and asthenia of the limb occurs. Sensory loss is the main clinical symptom. Some patient may experience tingling, burning pain , shooting pain down the legs. Neuropathic pain is nocturnal and causes insomnia[11]. They may develop postural hypotension, depressive symptoms [12]. A paradoxical feature is that both pain and numbness may co-exist in the same limb, a phenomenon aptly named as 'painful, painless' leg[13]. The former is due to C fibre damage and the latter due to A fiber damage[14].The commonest clinical presentation is reduced sense of vibration in the toes. Ankle jerk is absent and with advancement of disease knee reflex also gets involved. Capillary circulation is an important component, the alteration of which, can lead to neuropathy. Vasa Vasorum involvement hinders the clearance of metabolic end products from the tissue and also prevents nutrient delivery, thereby producing nerve damage [18]. Autonomic neuropathy leads to hypohidrosis and predisposes to breakage of dry skin[5].The

'purely'neuropathic foot is actually warm because of abnormal A-V shunting [16,17].This abnormal increase in blood flow can lead to neuropathic oedema. The American Diabetologist Association recommends screening for neuropathy ,at the time of diagnosis and for autonomic neuropathy five years after initial diagnosis in Type 1 and at the time of diagnosis in Type 2 Diabetes followed by annual screening thereafter.

Diabetic polyradiculopathy is a neurological manifestation characterized by disabling pain along the course of one or more nerve roots. Truncal radiculopathy causes pain over the thorax and abdomen. Involvement of the lumbar plexus or femoral nerve may cause severe pain in the thigh or hip and have diabetic amyotrophy. All these are usually self-limiting and resolve over 6-12 months.

Mononeuropathy is less frequently seen compared to polyneuropathy. It presents with pain and paresis along the distribution of a particular nerve. Involvement of Occulomotor Nerve is most common, heralded by diplopia. Examination shows ptosis, opthalmoplegia with normal pupil reaction to light. 4^{th} , 6^{th} and 7^{th} cranial nerves may be affected. Peripheral mononeuropathies with concomitant affliction of more than one nerves may occur.

Autonomic neuropathy can involve multiple systems. Cardiovasular effects include tachycardia at rest and orthostatic hypotension. Gastroparesis and bladder voiding abnormalities may be seen. Diabetic cystopathy is the

inability to sense bladder fullness and failure to empty the bladder completely. It can cause impotence and sexual dysfunction in both male and female. Increased sweating of the upper extremities and anhydrosis of the lower extremities may occur. Anhydrosis leads drying and crackling of feet which increases chances of ulcer formation. Peripheral neuropathy plays a key role in the events leading on to amputation.

PROPOSED HYPOTHESIS OF DIABETIC NEUROLOGICAL DAMAGE

- Nerve Ischaemia
- Protein kinase c activation
- Free radical injury
- Metabolic pathway hyperactivity eg. Polyol pathway
- Nerve Regeneration abnormalities
- Chronic High circulating glucose levels

CHARCOT FOOT (Ref Image 7)

Charcot neuroarthropathy is manifested by joint dislocations without major trauma and pathological fractures[19].It is of idiopathic origin. The concept by Virchow states that bony changes were due to unperceived sub-clinical trauma that are usually not noticed due to insensitivity of the joint. There is

reduced bone density in the foot in patients with Charcot neuropathy. The earliest clinical manifestation is swelling of the foot with pain or discomfort. The study by Boykao et al, found relationship between ulcer and charcot deformity, but other foot deformities were not independent ulcer predictors[20]. Acute Charcot foot may be mistaken for gout, cellulitis, and osteomyelitis[21]. Plain radiographs will show bone and joint destruction and loose bodies. Three phase ^{99m}Tc bisphosphonate demonstrates active Charcot process. The treatment given to Charcot foot patients are prolonged immobilization using total contact cast, (Ref Fig 8) Charcot restraint orthotic walker(CROW), Schotchcast boot(SCB),and Pneumatic walking braces.

ANGIOPATHY

Diabetes can affect both macro and microcirculation. In patients with Diabetes, atherosclerosis develops at an early age. Medial calcification, Diffuse intimal fibrosis and Atherosclerosis are the most common macrovascular changes observed with Diabetes. The most common risk factors associated to vascular component are dyslipidemia, hypertension, duration of Diabetes, severity of the disease, smoking, Insulin resistance. Moss and colleagues said that current smokers less than 30 years of age were more prone to ulcerate[22]. Cessation of smoking is associated with a decrease the atherogenic process. Hypertension is almost twice common in diabetics compared to non-diabetics.

Arteriosclerosis, specific diabetic microangiopathy and diabetic fibrillosis are the micro vascular changes observed with Diabetes. The typical histological changes are thickening of capillary basement membrane, proliferative changes in arterioles and arteries which include enlargement and proliferation of endothelial cells. Enlargement of endothelial cell is a feature in diabetes leading to small vessel occlusion, causing foot ulceration[23] termed 'small vessel disease' with the presence of palpable pulses in the foot. Increased resting blood flow due to denervated sympathetics causing loss of vasoconstriction, with loss of regulation in circulation in the arterio-venous vessels. A 'capillary steal' phenomenon is induced leading to shunting of blood away from the capillaries leading to reduced skin nutrition. This explains paradoxical ulceration despite increased blood flow.

PERIPHERAL VASCULAR DISEASE

Peripheral vascular disease occurs at an early age in diabetic patients . It is highly likely to involve vessels below popliteal artery. The mechanism by which vascular disease causing lesion of nerves are ischemia caused by occlusion of vessels, altered permeability of capillaries causing osmotic and metabolic derangements. In western countries, vascular alterations is an important factor for foot ulcerations causing major amputations later [24]. Minor trauma and antecedent infections increase blood requirement beyond the capacity, leading to ischemia and ulceration. Patients presents with intermittent

claudication, rest pain and nocturnal pain. Nocturnal pain and rest pain are relieved by keeping legs in dependent position. The circulation is predominantly caters to the splanchnic area during sleep, resulting in decreased perfusion of the lower extremities resulting in ischemic neuritis that disturbs sleep. The features of the ischemic limb are cold feet with absent pulses, delayed venous filling with blanching on elevation. There is loss of hair, thickened nails, and the skin appears shiny. Clinical assessment of the peripheral circulation is extremely useful in the assessment of outcome[25].

FAULTY WOUND HEALING

- Due to prolonged persistence of the abscess
- Poor granuloma formation
- Presence of bullae, necrobiosis
- Fungal infection of the nail
- High rate of carriage of staph.aureus in the nares

METABOLIC DERANGEMENT

Hyperglycemia in diabetics impair the complement fixation, ketosis impairs the leukocyte function, monocyte mediated immune functions are diminished, and alteration in polymorphonuclear leucocyte function leading to

deficient wound healing. Abnormal glucose levels and toxic metabolites play a role.

HAEMATOLOGY

Plasma viscosity, Platelet activity, Haemotocrit , red cells and white blood cells deformability are altered in diabetic patients. These changes profoundly influence the ischaemic process.

INFECTION

Infections in the foot are common with diabetics. Uncontrolled infection may progress to amputation[26]. Even with advancement in the treatment, uncontrolled sepsis cause about 60% of the lower extremity amputation [27,28]. In patients with gangrene due to diabetes amputations is the treatment[29]. Moist gangrene is most common in diabetic patients. Diabetic patients without infection, the prognosis is better than patients with infection. If the bones are involved, the risk of amputation is around eight times than those with involvement of soft tissue alone[30]. Fungal infections are common in the web space in the diabetics. Infections may occur in the nailbed. Preventing cross infection influences the outcome.

The descriptions of the involved region is by

- Severity
- Extent of the involvement (Ref image 9)
- Clinical appearance

- Location
- Etiology

There are two categories:

- Non-limb threatening
- Limb threatening
- Extensive infections that threaten limb or life require prompt hospitalization and appropriate treatment.

Foot infections occur at the site of trauma or ulceration[1]. If there is a breach in the epidermis, colonisation of the bacteria occur in the dermis and the underlying tissues. Usually the inflammatory signs are absent around the wound. They may present with symptoms like fever, nausea, and fatigue. They may present with unexplained increase in the blood sugar levels. If these features are found, sepsis should be suspected.

The lesions should be thoroughly examined to rule out exposed bones, joints and tendon sheaths. Failing to do so will result in rapid progression and involvement of deeper structures. The spread through tendon sheaths occurs both proximally and distally. Gold standard for diagnosing ulcer with infection is punch or tissue biopsy since the culture of the wound are often misleading and may not represent the organism within the underlying granulation tissue. The severity of the infections are assessed by

- analyzing depth of the wound,
- presence of ischemia,
- the presence of infection

In the presence of osteomyelitis, bone sampling and sensitivity testing become minimum required procedures to prevent progression to more aggressive stages of the disease.

FOOT ULCER DEFINITION & CLASSIFICATIONS OF DIABETIC FOOT

The International consensus currently defines a " foot ulcer" in the diabetic patients as a "full-thickness wound below the ankle , irrespective of the duration" [31].

Classifications:

The International Working Group On Diabetic Foot recommends use of a uniform classification system to

- Enable universal understanding among health care providers
- Provide accurate assessement of healing potential
- Standardize management protocols
- Eliminate observer variations
- Should be universally acceptable and usable[32].

Universally used classification is WAGNER'S CLASSIFICATION[33]

Diabetics possess a life time risk of around 15% for possible ulcer formation[34].

WAGNER CLASSIFICATION[36]

Grade 0 - No ulcer but high risk foot (deformity or cellulitis)

1- superficial diabetic ulcer

2 - ulcer extends to ligaments, tendons, joints, capsule or deep fascia without abscess and /or osteomyelitis

3 – deep ulcer with abscess/ osteomyelitis/ joint sepsis

4 - gangrene localized to portion of fore foot

5 - extensive gangrenous involvement of entire foot

This most valuable grading for the diabetic ulcer foot designed by William Wagner. It is also known as WAGNER-MEGITT'S CLASSIFICATION. This system help to analyze the progress of the patient, both positive and negative outcomes, and to standardize the treatment plan.

CLASSIFICATION – UNIVERSITY OF TEXAS[38]

Aetiology (Stage) included -

Stages

–Stage A: No infection or ischemia

–Stage B: Infection present

–Stage C: Ischaemia present

–Stage D: Infection and ischaemia present.

Grading

–Grade 0: Epithelialized wound

–Grade 1: Superficial wound

–Grade 2: Wound penetrates to tendon or capsule

–Grade 3: Wound penetrates to bone or joint

The other classification is **SAD system**, which adds to the Texas system the cross-sectional area and the presence of neuropathy or not [35].

The 'PEDIS' system includes

- Perfusion (ischaemia)
- Extent
- Depth
- Infection
- Sensation (neuropathy)

More recently, International Working Group on Diabetic Foot (IWGDF) has created a classification that grades wound size ,perfusion of foot, the presence of infection, and the presence of sensation.The flaw in all classifications is that none of these systems considers the duration of the foot ulcer[37], which has great bearing on healing [38].

ASSESSMENT OF DIABETIC ULCER FOOT

Proper assessment of diabetic foot involves adequate history, clinical examination and investigations.

HISTORY

- MEDICAL HISTORY
    1. Diabetes – Duration
    2. Treatment history
    3. Co-morbidities
    3. Nutrition
    4. Addictions
    5. Current drug intake
    6. Hypersensitivities
    7. Past Medical or surgical history
- GENERAL HISTORY
    1. Everyday activities
    2. Foot Protection
    3. Callus formation
    4. Bony deformities of the foot
    5. Neuropathic symptoms
    6. Claudication or rest pain

- WOUND/ ULCER HISTORY

    1. Area involved
    2. Number of months involved
    3. Precipitating cause
    4. Previous involvement in the same site
    5. Infection
    6. Hospitalization
    7. Wound care
    8. Wound healing
    9. Patient adherence to advice
    10. Social problems hampering adequate wound care
    11. Previous foot trauma or surgery
    12. Presence of pedal edema
    13. Charcot arthroneuropathy

EXAMINATION

1. skin changes

2. sweating

3. infection: clinical assessment is useful. One should look for

- The discharge whether serous or purulent,
- soft tissue involvement

- presence of temperature
- pus culture and sensitivity

4. bony changes

5. Deformities of the foot

6. wasting of small muscle

7.Surface temperature of skin

LABORATORY TESTS

This includes fasting or random glucose measurement, glycosylated haemoglobin, complete blood count, C - reactive protein, and urinalysis.

Neurological assessment

- Tuning fork assessment of vibration sense
- C-Fibre assessment by pinprick.
- 10-g monofilament to assess the foot ulcer risk status[39], a semi-quantitative measure of light touch[40,41]. (Ref Image 10)
- Light touch assessment by using cotton wisp

Vascular assessment

- Peripheral pedal pulses should be palpated
- Doppler ultrasound probe evaluation

- Ankle-Brachial index. An ankle pressure of less than 70 mm hg leads to poor healing of ulcers; if it is more than 100mm Hg, the prognosis is good.
- Transcutaneous oxygen tension ,a noninvasive test of circulation in the periphery.
- Skin blood flow calculated from Xenon 133 clearance.
- Segmental arterial pressures, waveform analysis by using Doppler

INDICES OF POOR WOUND HEALING

If the ankle-brachial index of less than 0.8, the measured toe blood pressures less than 40 mmHg, or the level of transcutaneous oxygen tension ($TcPo_2$) less than 30 mmHg are associated with poor healing of the wound. If these parameters are present, vascular surgeon consultation becomes essential.

Quantitative sensory testing

Vibration assessment

Biothesiometer, (Ref Image 11) Neurothesiometer –handheld device to assess vibration perception. It is portable and easy to use in an out-patient setting. Detection of diminished vibration sense is a predictor of impending ulceration

Temperature assessment

A portable instrument Neuroquick [43], useful for testing cold sensation, and for screening early neuropathy.

Infection assessment

By doing pus culture and sensitivity. It is often misleading, not represent the organism underlying the granulation tissue. " Gold standard" test is punch or tissue biopsy.

Repetitive Moderate stress assessment

Thermography- Patients with insensitive feet shows hyperemic thermographic pattern.

"In-shoe" foot prints helps to detect the area of maximum stress.

Osteopathy assessment

To confirm the suspected cases of the bone involvement imaging studies are usually done. It is also useful to plan the correct treatment for the affected foot.

Plain X-rays

Relatively cheap, quick, widely available. It is the first imaging modality for patients with suspected bone infection. It takes two weeks for the changes to be seen in the plain x-ray. In plain x-ray, presence of calcified vessels, gas, foreign body, osteolytic changes, fractures, dislocations in

neuropathic arthropathy, biomechanical alterations and osteomyelitic changes can be seen. The osteomyelitic changes are focal osteopenia, cortical erosion, and periosteal reaction, soft tissue swelling and permeative radiolucency . It is very difficult to differentiate aseptic neuroarthropathy by means of plain x-ray from suspected osteomyelitis. Even if the initial images on plain x-rays are not contributory to the diagnosis of suspected osteomyelitis, regular radiologic imges should be done.

Direct Digital Radiography markedly reduces the effect of radiographic overexposure. Routine views are dorsi-plantar and oblique views. Specialized views are lateral view, standing view without support, seasomoids, forefoot, subtalar, heel and ankle views. Currently weight bearing dynamic views are recommended.

LIMITATIONS OF RADIOGRAPHY

- Difficult to assess the soft tissue involvement
- Due to the presence of overlying structures, not accurately measures bony involvement
- Only around the bony edges, cortical bone involvement is made out

MAGNATIC RESONANCE IMAGING

It is the best modality to delineate infection and neuropathy with its precision reaching almost 100%. Presence of fluid in the soft tissue can be made out. To know the extent of involvement, this modality is most useful[43].

ANATOMY OF THE SOLE OF THE FOOT

SKIN

It is thick , firmly attached to the deep fascia by fibrous bands. sweat glands are present in large numbers. The sensory nerve supply to the medial skin of sole is derived from the tibial nerve, lateral part of the sole is innervated by corresponding plantar nerve,and its Medial two thirds is innervated by medial plantar nerve.

DEEP FASCIA

Deep treasure metatarsal ligaments and aponeurosis together constitute deep fascia. Flexor retinaculam extends from the Medial malleolus to the medial surface of calcaneum. It attaches the tendons of deep muscles to the medially in the ankle. The aponeurosis occupies the central area of Sole, triangular in shape and its apex is attached to the calcaneum, and its base attached to the toes. Each slip has two bands, passing superficially to the skin and deeply to the root of toe. Fibrous septa form the fascial spaces of sole,

gives firm attachment to the overlying skin. It protects the underlying vessels, tendons, nerves. It helps to maintain the arches in the foot.

MUSCLES OF THE SOLE

They are divided into four layers.

I Layer Abductor hallusis

Abductor digiti minimi

Flexor digitorum brevis

II Layer Lumbricals

Flexor hallucis longus

Flexor digitorum longus

III Layer Flexor hallusis brevis

Flexor digiti minimi brevis

Adductor hallusis

IV Layer Interossei

Tibialis posterior

Peroneus longus

ARTERIAL SUPPLY

1.Medial Plantar Artery

Medial side of toe is supplied by it, gives off numerous cutaneous, muscular and articular branches.

2.Lateral Plantar Artery

Plantar arch is supplied by it. Plantar arch gives digital arteries to lateral side of the small toe and both sides of four lateral toes.

3.Dorsalis Pedis Artery(DPA)

DPA joins with the plantar artery on the lateral aspect and supplies plantar arch .

Its branches are

-Lateral tarsal artery

-First dorsal metatarsal artery which supplies both side of big toe.

-Arcuate artery supplies metatarsal branches to the toes.

Medial and lateral plantar veins accompany the corresponding arteries and join behind the medial malleolus to form the posterior tibial venae commitanes.

NERVE SUPPLY

1. Lateral Plantar Nerve

Supplies flexor digit minimi, abductor hallucis, quadaratus, plantaris, abductor digiti minimi,second and third and fourth lumbricals and all interossei.It supplies cutaneous twigs to the skin sole on lateral aspect, one and half toes on the lateral aspect,and its nail beds and tips.

2.Medial Plantar Nerve

supplies muscular branches to abductor hallusis, flexor hallusis brevis,flexor digitorum brevis. It also supplies cutaneous branches to medial three and half toes and its corresponding nail beds and tips.

DORSUM OF THE FOOT

SKIN

Skin on the dorsal aspect is freely mobile on the underlying tendons and bones. Sensory nerve supply to the dorsum is derived from the superficial peroneal nerve. It also receives sensory innervations by deep peroneal,saphenous, sural nerves.The skin covering the dorsal surfaces of terminal phalanges, nail beds are supplied by medial and lateral plantar nerves.

Knowledge of the spaces of the foot is very important because infections in any of the spaces result in extension along fascia or tendon. The four medial spaces can be approached from inside of the foot by an incision along inner border of

first metatarsal bone. The digital vessels arise from dorsal arch pass via intermetatarsal ligaments to the toes. Infection of the toe or web space if not adequately controlled spreads deeper. It reaches the tendon sheaths of long flexors or lumbricals and spread to 3rd layer. Very high pressure build up in the closed area because of pus, edema, and presence of gas forming organisms. This causes tissue necrosis and mechanical pressure on digital vessels leads to gangrene of the foot.

ANATOMY OF THE LEG

Superficial fascia

Contains superficial veins like great saphenous vein, short saphenous vein, cutaneous nerves like infrapatellar branch of saphenous nerve, saphenous nerve,lateral cutaneous nerve of calf, superficial peroneal nerve, sural nerve, lymphatics, and small unnamed arteries.

Deep fascia

Extension of deep fascia form the septa divide the leg into three compartments anterior, posterior and lateral.

Anterior compartment

Muscles

Tibialis anterior

Extensor hallucis longus

Extensor digitorum longus

Peroneus tertius

Vessels Anterior Tibial vessels

Nerve Deep peroneal nerve

Lateral Compartment

Muscles

Peroneus longus

Peroneus brevis

Nerve Superficial peroneal nerve

Vessels Peroneal vessels

MEDIAL SIDE OF THE LEG

Formed by medial surface of the shaft of tibia. The greater part of this surface is subcutaneous and is covered by skin and superficial fascia. Three muscles are inserted into the upper part of medial surface of the tibia from three compartments of the thigh namely Sartorius, gracilis, and semitendinosus forming Guy ropes.

BACK OF THE LEG

Superficial fascia of the back of the leg contains small and great saphenous veins and their tributaries, several cutaneous nerves,and medial and lateral calcaneal arteries.

Superficial muscles of this area are

- Gastronemius
- Soleus
- Plantaris

Nerve supply to superficial muscles of the back is Tibial nerve.

Posterior group of muscles that are present in deep aspect are

- Popliteus
- Flexor digitorum longus
- Flexor hallucis longus
- Tibialis posterior

Vascular supply to this area by Posterior tibial vessels.

ANATOMY OF THE THIGH

FRONT OF THE THIGH

The superficial fascia of the front of the thigh contains great saphenous vein, cutaneous nerves, vessels, lymphatics and lymph nodes.The upper third of the

thigh medially contains the femoral triangle, middle third carries the femoral vessels through the adductor canal. Muscles of the frontal aspect of the thigh are

Sartorius

- Rectus femoris
- Vastus lateralis
- Vastus intermedius
- Vastus medialis

Nerve supply : Femoral Nerve

MEDIAL ASPECT OF THE THIGH

Muscles

Adductor longus

Adductor brevis

Adductor magnus

Gracilis

Pectineus

Obturator externus

Nerve supply

Obturator nerve

Accessory obturator nerve

Arterial supply

Obturator artery

Medial circumflex femoral artery

BACK OF THE THIGH

Muscles

Semitendinosus

Semimembranosus

Biceps femoris

Nerve supply Sciatic nerve

Vascular supply

Lateral circumflex femoral

Medial circumflex femoral vessels

BIOMECHANICS OF FOOT

Biomechanics is a science that deals with effect of forces acting on living tissues. The major cause of ulcer formation in diabetics is loss of pain sensitivity.[44] Most of the non-healing ulcers are not caused by super-added medical conditions, but due to presence of basic biomechanical factors. Progressive elevation of plantar pressure is a reliable predictor of ulcer formation[45,46].Biomechanical factors play pivotal role in all phases of

management of foot ulcers starting from prevention level to tertiary expert care level.

STRESS AND STRESS CONCENTRATION

The mean pressure acting at the feet of a 100kg individual is about 75 kilopascals. Dynamic pressures are higher compared to static pressures and simple walking[47].Shearing stress also has a key part in the evolution of ulcers.

NEUROPATHY AND HIGH PRESSURE (Ref image 12)

Peripheral neuropathy results in loss of sensations that confer protection against injury. This loss is severe enough that patients may not even perceive severe trauma such as penetrating injury to the feet or even scalding caused by boiling water or burns. Disuse atrophy predisposes to ulceration. High pressure areas that frequently ulcerate include toes and head of metatarsals[50,51]. Pressure applied repeatedly over the same areas especially those overlying bony prominences in the setting of profound sensory loss causes ulcer formation that starts from within out ie, from bone to the outer soft tissue.[48.] Callus are often found on inspection. They have a dark base indicative of an underlying deep ulcer which bleeds and stains the deep surface of the callus[49].

INTRINSIC FACTORS CAUSING ELEVATED PRESSURE

- Changes in foot architecture like long second metatarsal

- Increased angle of foot arches
- Soft tissue lesions like callus, clawing of toes due to fat pad migration leaving bony prominences exposed
- Limited joint mobility

EXTRINSIC FACTORS CAUSING ELEVATED PRESSURE

- Poor foot wear
- Ill fitting shoes
- Non compliant soles
- Prior surgery

ACTIVITIES CAUSING ELEVATED PRESSURE

- Bare foot walking
- Improper shoes
- Improper foot care
- Altered gait

MANAGEMENT OF DIABETIC FOOT

The primary target of diabetic foot management is wound closure as early as possible. Enabling quick healing of ulcers and preventing reoccurrence reduces the need for amputations.

The essential avenues of diabetic foot management are:

- Control of co-morbidities
- Ensuring adequate vascularity
- Assessing psychosocial factors
- Ulcer apparaisal
- Ulcer bed preparation
- Relieving pressure

Treatment differs according to the nature of the wound. It can be either 'conservative' therapy or surgical intervention. Before planning for surgical intervention care should be given in the form of antibiotics, adequate care of the foot. Debridement of wound should be given. If necessary offloading techniques may be used for the wound to heal. Patient should be advised to maintain good glycaemic control. All the devitalized tissues should be removed. If there is involvement of the bone, it should be removed. If there is presence of pus, incision and through drainage should be done. These prompt measures may provide healing of the wound.

PAIN MANAGEMENT IN DIABETIC NEUROPATHY

Management components include

- Control of blood glucose levels
- Reduction of Cardiac risk factors. This encompasses control of blood pressure, Body mass index, Hypercholesterolemia and tobacco use.[52]

- Tricyclic anti-depressnats like Amitryptilin and SSRI group of drugs like Duloxetine are primarily used[53]. These drugs in their corresponding doses used have a safe side effect chances[54].
- Anticonvulsants are useful in the treatment of pain due to neurological involvement. The drugs commonly used are barbiturates and carbamazepine. The recent therapies are gabapentin[55], pregabalin[56]. These new drugs provide pain relief better than the previously used drugs.
- Opioid derivatives- These derivatives are useful in relieving neuralgia[57].
- Topical capsaicin- These act via reduction in levels of Substance P. The draw back is that there may be an initial increase in pain before drug effect takes action[58].
- Alpha-lipoic acid Infusion - 600 mg per day for 3-weeks is helpful in neuropathic pain[59].
- Isosorbide Dinitrate Spray and Glyceryl Trinitrate Patch-
- Nitrate spray[60] and patch[61] applications lead to improvement of neuropathic pain.
- Non-pharmacological treatments
    1. Acupuncture
    2. Near-infrared therapy
    3. Low-intensity laser therapy[62]
    4. Transcutaneous electrical stimulation

5. Direct stimulation of the spinal cord by surgical implants
6. Frequency modulated electromagnetic neural stimulation[63].

MANAGEMENT OF PERIPHERAL VASCULAR DISEASE

Before planning for surgical intervention in the form of either bypass or angioplasty ,it should be analysed whether this procedure will actually benefit the patient, the outcome of the planned surgery, and complications associated with the planned surgery either immediate and long term. If the vascular surgeon is competent to do the endovascular surgeries, this form of treatment is preferred because of minimum morbidity and in the expert hands this is very safe procedure.

Indications for Angioplasty:

- Short stenoses
- Aorto-illiacs dilatable
- Life expectancy less than 2 years
- Occlusions less than 20cm
- Ulceration due to Venous causes
- ABPI > 0.5

Indications for surgical bypass:

- Femoral Artery block
- Advanced ischaemic necrosis
- ABPI < 0.5
- Life expectancy > 2 years
- Combined inflow and CFA disease

Absolute indications for surgery:

- Unsuccessful angioplasty
- Rutherford Class II ischaemia
- Aneurysms
- Extensive calcification

When the above options are not available or if the patients are not affordable to these procedures then palliative approach is the best option. Amputations are usually done for these patients. Rehabilitative measures should be given for these patients.

TREATMENT OF DIABETIC FOOT INFECTIONS

Treatment of diabetic foot infections are divided into non-limb threatening and threatening infections.

Non-limb threatening infections

To define, these are medically stable patients having no signs of systemic sepsis. They are ideal candidates for outpatient management under close supervision.

Clinically, these group of patients have superficial ulcers with minimal ischaemia and lack bone or joint involvement. Cellulitis is confined to within 2cm of the ulcer margins. Many of these cases are monomicrobial with staphylococcus aureus, staphylococcus epidermiditis and beta hemolytic streptococci being the most common ones to be isolated. Samples for cultures are acquired by curettage of the infected ulcer. Oral antibiotic therapy is initially given. MRSA should be covered under the spectrum. The ulcer should be debrided as often as necessary.

Limb-threatening infections

This group includes

- Cellulitis beyond 2cm of ulcer margin
- lymphangitis
- Ischemic Tissue necrosis
- Odour
- Gangrene

- Bone involvement

These patients require emergent hospital admission and appropriate intervention. The patient's co-morbidities should be evaluated. Extent of infection should be thoroughly assessed. A team approach in treatment improves outcomes.

Initial management by a surgeon includes debridement, drainage of toxic fluid or pus, decompression by fasciotomy or limited amputations to curtail progress of infection. During this, wide range of culture specimens should be obtained to identify bacteria and fungal growth. Sub-Arachnoid Blocks should be avoided in these patients.

The presence of hemodynamic imbalance or profound systemic compromise should not deter the surgeon from taking up debridement. Only clearance of infected tissue will improve the internal milieu of the patient. Most of such cases have poly-microbial infections requiring intravenous antibiotics initially rather than oral medication. Osteomyelitis should be evaluated radiologically and microbiologically. Debridement of the infected bone and even minor amputations may be needed. Bone cement containing antibiotics are available for use after adequate debridement, provided adequate soft tissue cover is availabe.

DRESSINGS

Dressings are done to keep the wound clean and free. It removes excess bacteria from the wound. It removes excess fluid. Materials used should not cause toxicity to the wound. It should be non-adhesive. Various types are

1. Honey dressing- provides moisture, decreases odour, reduce inflammation, reduce oedema and exudates.
2. Silver-containing dressings- Silver is having broad spectrum of activity. It kills yeast, fungi, viruses and methicillin and vancomycin strains[64].
3. Iodine-Dressings.
4. Hydrocolloid Dressings- Create moist healing environment, should not be used in heavily infected wound
5. Alginate dressings- Derived from sea weed, have the capacity to absorb large amounts of exudates.
6. Hydrogels- Promote autolytic wound debridement in dry wounds, absorb exudates in moist wounds.
7. Hyalofill- Derivative of hyaluronic acid, should be used on clean wounds.
8. Living skin equivalents- Dermagraft or graftskin.

LARVAL THERAPY

Maggots are living chemical factories. Worms remove dead tissues by the production of a mixture of proteolytic enzymes that breakdown dead tissue to a

semi-liquid form ingested by creatures, stimulate fibroblast growth in vitro, eliminate odour and reduce wound related pain.

GROWTH FACTORS

Growth factors play a key role, regulating all aspects of wound healing. Epidermal growth factor(EGF), fibroblast growth factor 2 (FGF-2) and platelet derived growth factor(PDGF) all have been approved in the treatment of diabetic foot ulcers.

OFFLOADING THERAPY

The choice of technique is based on the physical form and compliance with the treatment, and also site and severity of the ulcer. The aim is to reduce dynamic foot pressure[74].

Total contact cast (TCC) is gold standard in management. Total contact cast should not be used in patients with local sepsis and vascular compromise. Other options are Removable cast walkers, Charcot restraint orthotic walkers, Healing sandals and half shoes, felted foam padded dressings, therapeutic shoes and insoles.

This line of management should be continued till there is complete healing of ulcers.

SURGICAL INTERVENTION

It is often indicated for deep infection or for the treatment of recurrent or recalcitrant ulceration. They include debridement of recalcitrant or infected ulcers, drainage and debridement of soft tissue infection,bone resection for relief of pressure and or for osteomyelitis; closer techniques; Achilles lengthening surgery; and reconstruction of forefoot deformities. Primary or delayed closure is advocated as more effective, much more cost-efficient and successful. Low pressure or vacuum assisted closure is particularly valuable when there is insufficient soft tissue surplus to allow for primary closure of the wound edges.

DEBRIDEMENT

Debridement serves several purposes:

- Excision of dead tissues and callus.
- Pressure alleviation
- Ulcer bed assessment
- Probing of tracks and tunnels
- Decreasing microbiological load

There are Five types of debridement

- Surgical
- Enzymatic
- Autolytic

- Mechanical
- Biological

Early surgical intervention by debridement has been demonstrated to be the best.

SURGICAL DEBRIDEMENT

Aggressive removal of the devitalized tissues is done by sharp scissors and scalpels. Extent of debridement should be sufficient enough to reach all margins of infection both in the deeper plane and also in the horizontal plane. The aim of this approach is to convert a chronic non-healing wound into an actively bleeding acute wound that will have the capacity to granulate and regenerate. Deep seated abscess mandates hospitalisation and immediate incision and drainage. Removal of bones either locally or by limited amputations may be necessary. Repeated removal of necrotic tissue expedites rate of healing. This is known as as “maintenance debridement”.

Hydro surgery

Its properties are

- Precise and
- Limited Excision
- Negligible thermal damage to the tissues.

An extremely painful wound may benefit from enzymatic debridement.

Vascular wounds are candidates for enzymatic debridement.

MOISTURE BALANCE

Moisture accelerates re-epithelization in a wound. Tissue moisture is a term used to convey the importance of keeping wounds moist and free of excess fluids. A moist wound environment promotes granulation and autolytic processes. Effective management of chronic wound fluids is an essential part of wound bed preparation. It also helps in addressing the issues of cellular dysfunction and biochemical imbalance.

ADVANCES IN WOUND CARE

Preparation of ulcer bed enables removing barriers which hinder healing and to start the healing process. Advanced care sometimes becomes the only means of attaining wound closure. The discovery of recombinant growth factors, genetic manipulation, artificial tissues, stem cell therapy have empowered the surgeon and wound-care provider to aid in angiogenesis to accelerate healing.

VAC THERAPY

Delivery of intermittent or continuous sub-atmospheric pressure through a specialized pump connected to open-celled foam surface dressing covered

with an adhesive drape to maintain a closed environment. It increses blood flow, decreases local tissue edema, removes excessive fluid and pro-inflammatory exudates from the wound bed[65,66].

AMPUTATIONS

Amputations, an unpleasant but often final end result of the diabetic foot. It is performed for multiple reasons and can be either curative or emergent. Amputation level selection aims at achieving balance between preservation of limb length and function with the ability of the wound to heal properly.

Currently available vascular surgical advances have made 'limb sparing' more and more feasible. Endovascular restoration of vascularity have made it possible to do more distal amputations. Pre amputation vascular intervention must be done to limit level of amputation and also to facilitate proper stump healing.

AIM OF DECIDING LEVEL OF AMPUTATION

- To leave behind a stump that can readily accept prosthetic shoe, orthotic device or complete limb prosthesis
- To create a stump that is less likely to breakdown from external pressures
- To prevent dynamic imbalances that may occur due to migration of digits such as in migration of the other digits after 1st MTP joint disarticulation,

varus deformity that occurs due to lateral loading of the foot following 5th Toe Ray amputation.

- To facilitate primary wound healing so as to enable rapid healing of the stump and early rehabilitation of the amputee.

If infections are not controlled or due to the advancement in the disease process, diabetics usually succumb to lower extremity amputation. The incidence of amputations for non-traumatic etiologies is ten times higher in Diabetes[67,68]. Costs of amputation and its descendent managements are very high[69] This is due to length of hospitalization that is required and due to multiple investigations and repeated surgical and vascular interventions that may be required.

Various amputations of lower extremity are

- Ray's amputation
- transmetatarsal (Gillies)
- tarsometatarsal (lisfranc's)
- midtarsal(chopart's)
- syme's
- Below-knee(Burgess)
- Transcondylar
- above-knee

The three most common indications for major lower extremity amputations are

- acute limb ischaemia
- chronic critical limb ischaemia
- major infection due to malperforans ulcers in diabetics with normal arterial circulation.

GOALS

The goals of major lower extremity amputations are :

- to eliminate the nonviable tissue
- to provide a stump with best chance to heal
- to provide a stump with best chance of long term function-ambulation with prosthesis.

IDEAL STUMP

- The ideal stump should heal adequately
- should have rounded ,gentle contour with adequate muscle padding
- should have adequate length to bear prosthesis
- should have thin scar which does not interfere with prosthetic function
- should have adequate joint movement
- should have adequate blood supply.

GENERAL PRINCIPLES APPLICABLE TO AMPUTATION SURGERY

SKIN

Flaps should be sutured in a tension free manner and the scar should be well healed and non-adherent to the bone.

MUSCLE

Myodesis should be performed to facilitate balanced action of opposing muscle groups.

NERVE

Neuroma formation should be prevented by dividing the nerve at a higher level by applying adequate traction and allowing it to retract into the stump under cover of muscles. Nerves should never be ligated.

BLOOD VESSELS

Visible bleeding alone does not indicate optimum level of amputation.[70] Wound healing in reality is dependent on micro-circulation. Vessels must be suture ligated, arteries and veins in separate group to avoid iatrogenic AV fistula formation.

BONES

Bone should be cut at a higher level and ends beveled so as to avoid protruding bone that will interfere with healing of stump and also result in a painful end bearing stump.

STUMP DRESSINGS

A cotton wool followed by crepe bandage is commonly used dressing for the amputation stump. A rigid cast support [71] enables wound protection, contracture prevention and oedema reduction.

TYPES OF AMPUTATION

RAY amputation

Amputation of the toe with the head of metatarsal or metacarpals.

TRANSMETATARSAL AMPUTATION (GILLIES')

Amputation is done proximal to the neck of the metatarsals, distal to the base.

LISFRANC'S AMPUTATION (TARSOMETATARSAL)

Here tarsometatarsal joint is disarticulated with a long volar flap.

CHOPART'S AMPUTATION (MIDTARSAL)

Here talonavicular and calcaneocuboid joints are disarticulated. Tibialis anterior is sutured to the drilled talus bone. A long volar flap is used and immobilized for six weeks after surgery.

SYME'S AMPUTATION

It is removal of the foot with calcaneum and cutting tibia and fibula just above the ankle joint with retaining heel flap (dividing both malleoli).Heel flap

is supplied by medial and lateral calcaneal vessels . Elephant boot is used for the limb after syme's amputation. Many patients walk well with syme's stump without difficulty. It is presently mainly used in trauma(crush injuries) and malignancies of the distal part of the foot.

PIROGOFF'S AMPUTATION

It is like syme's amputation except the posterior part of the calcaneum is retained along with heel flap. It provides longer stump than syme's amputation.

TRANSTIBIAL(BELOW-KNEE) AMPUTATION (Ref image 13)

Knee joint is spared. The ideal stump is 15cms long

The advantages of preserving the knee joint are

- lower kinetic energy requirement
- near normal gait
- Ease of using prosthesis
- Self Sufficiency and reduced dependancy
- Quicker rehabilitation
- Less expensive prosthesis

KNEE DISARTICULATION(THROUGH-KNEE) AMPUTATION

It is through the joint and does not disturb the bone. It is used in patients with poor general condition and those who are not amenable to prosthetic mobilization

TRANSFEMORAL(ABOVE-KNEE) AMPUTATION (Ref image 14)

About 12-15cm of lower end of femur should be removed. Usually equal anterior and posterior flaps are used. If femur length less than 10cms this procedure is not possible. If femur length is less than 10 cms, then should proceed with hip disarticulation. The marked reduction in limb length drastically reduces propulsive power and manipulation of the prosthesis. Efficient ambulation depends solely on the user's ability to mobilize the artificial knee joint in the prosthesis.

CURATIVE VERSUS EMERGENT SURGERY

Performance of amputation in the elective setting may not always be a possibility. When serious infections such as gas gangrene are starting to set in, it becomes mandatory to perform an emergency amputation. Before surgical intervention, pre-existing infection should be dealt with. Elective amputations are usually curative ie, primary wound healing is facilitated by raising flaps and closing the wound primarily. Emergency amputations aim at removal of

necrotic tissue only and not at healing the stump primarily. Subsequent surgery may be required to close the wound once the infection has been controlled.

COMPLICATIONS OF AMPUTATION SURGERY

Early complications:

- Hemorrhage
- Infection
- Haematoma

Late complications:

- Pain
- Flap necrosis
- Ring sequestrum formation
- Ulceration of the stump
- Painful scar
- Phantom limb

POSTOPERATIVE PERIOD AFTER AMPUTATION

- Regular physiotherapy
- Regular dressing
- Crutch is used initially
- After 3 months prosthesis is used
- Rehabilitation

PROBLEMS IN AMPUTEES IN DIABETICS

Fluid overload caused by cardiac failure or renal diseases in diabetics may cause fluctuation in the volume of the stump. This provides challenges in proper fitting and maintainance of prosthesis.

FOOT WEAR & DIABETES

The state of art in footwear prescription for patients with Diabetes is still rudimentary. Narrow shoes, pressure from footwear, inadequate foot wear, trauma from foot wear, barefoot walking, are widely believed to cause many foot injuries and even amputations in persons with Diabetes.

Therapeutic foot wear works by

- Accomodating the bony deformity
- Adequate space for allowing use of thick special insoles etc.
- Dynamic load reduction
- Load transference from risk areas
- Forcing the patient to acquire a proper gait and stance
- Providing pads to off-load the metatarsals
- Providing medial longitudinal arch support

- Newly detected diabetic patients should be educated about the need for appropriate foot wear.
- For diabetic patients who is at risk for an ulcer should use extra-depth shoes[72]
- Delayed institution can cause reulceration
- For patients with a recently healed ulcer should be given well-cushioned walking splint or orthopedic walker so that fragile tissue can heal
- Graduated use of the new foot wear should be advocated
- For charcot patient, patellar tendon bearing brace to transfer some of the load to the leg.

OBSERVATIONS AND RESULTS

Risk factors associated with MAJOR amputation in diabetic foot

1) AGE

Table1: Table showing age distribution of study subjects

Age group (years)	Number of subjects
20-30	4
30-40	6
40-50	10
50-60	26
60-70	20
70-80	12
80-90	3
TOTAL	**81**

Amputations were more common in the 50-70 year age group.

2) SEX

Table 2: Among the study subjects, 48(59.5%) were male and 33(40.5%) were female.

Sex	Number of subjects
Male	48
Female	33
Total	**81**

Amputations were more common males compared to females.

3) DURATION OF DIABETES MELLITUS

TABLE3: Table showing duration of Diabetes Mellitus in the study subjects

DURATION OF DIABETES	Number of subjects
<5YEARS	11
6-10 YEARS	25
11-20 YEARS	31
>20 YEARS	14
TOTAL	**81**

The actual duration of Diabetes Mellitus may be underestimated due to delay in diagnosis.

4)Previous history of minor amputation

Table showing previous history of minor amputation of study subjects

Previous history of minor amputation	**Number of subjects**
Nil	42
Toe disarticulation	14
Ray amputation	18
Mid tarsal/Tarso metatarsal	5
Syme's Amputaion	2
TOTAL	**81**

Those who underwent previous minor amputation of the lower extremity are more prone for major amputation of the lower extremity.

5) Educational Status

Majority of the subjects had primary school education or less.

Table showing educational status of study subjects

Educational Status	**Number of subjects**
Illiterate	14
Primary school	48
Higher Secondary	13
Degree or higher	6
TOTAL	**81**

6) Use of footwear

Use of Foot wear	**Number of subjects**
Yes	53
No	28
TOTAL	**81**

Among the study subjects 53 (65.4%) were in the habit of using footwear while 28 (34.6%) were not.

7)Smoking

Smoker	Number of subjects
Yes	34
No	47
TOTAL	**81**

34(42%) of the study subjects were smokers while 47(58%) were non smokers

8) Arterial insufficiency in the affected limb

The arterial status of the affected limb was assessed by recording the Ankle brachial index.

ABPI	Number of subjects
<0.3	12
0.4-0.8	32
>0.9	27
TOTAL	**71**

Ankle brachial pressure could be measured in only 71 of the 81 patients prior to the amputation.

9) Neuropathy

Neuropathy	Number of subjects
Present	62
Absent	19
TOTAL	**81**

The presence of neuropathy was assessed by Tuning fork testing and biothesiometry.

Among the subjects studied 62(76.5%) had neuropathy while 19(23.5%) did not have neuropathy.

10) Anemia

Hemoglobin (g/dl)	Number of subjects
<8	39
8-12	28
>12	14
TOTAL	**81**

67 (82%) of the 81 patients studied had a haemoglobin level of <12 gm%

11) RBS value on admission

RBS (mg/dl)	Number of subjects
<110	12
110-140	17
140-200	7
>200	45
TOTAL	**81**

45 (55.55%) of the subjects had RBS value more than 200 mg% on admission.

12) Renal function test

Sr.Creatinine (mg/dl)	Number of subjects
<1.0	57
>1.0	24
TOTAL	**81**

24(29.6%) out of the 81 subjects had elevated renal function test (S. creatinine>1.0 mg/dl)

13) Serum albumin levels

S. Albumin(g/dl)	Number of subjects
>3.0	13
2-3	45
<2	23
TOTAL	**81**

14) Presence of osteomyelitis

Osteomyelitic changes	Number of subjects
Present	29
Absent	52
TOTAL	**81**

Among the study subjects 29(35.8%) had evidence of osteomyelitis on imaging.

15) Pus culture and sensitivity

MICROORGANISM	**Number of subjects**
Proteus vulgaris	9
Escherichia coli	15
Pseudomonas aeruginosa	18
Staphyococcus aureus	13
Polymicrobial	26
TOTAL	**81**

Polymicrobial infections were the most common among the study subjects.

DISCUSSION

The studies by Most RS et al [75], Siitonen OI et al [76], Armstrong DJ et al [77], Group TG [78], have shown increasing age and male gender are unavoidable risk factors for amputation. In our study amputations were more common in the Fifth and sixth decade and there is a male preponderance of 1.46:1.

Kumar [79], Walters [80] have found that increasing duration of the diabetes is a risk factor for amputation. In our study, majority of the patients had long duration of diabetes.

Studies by Litzelman et al [81] , Kumar et al , Carrington et al, Boyko et al, Abbott et al, Kastenbauer et al, have found previous history of amputation is a risk factor for amputation. In our study, 44.4% of subjects has a previous history of minor amputation.

In our study, 76.5% had primary schooling or lesser education. This is quite low compared to western studies. If the educational status is low, they are usually unaware of preventive steps to be taken. Attendance of health care facilities is likely to be less frequent(Adams AS et al [82]).

Studies by ChantelauE et al [83] have found the use of footwear in diabetes mellitus prevents the development of the initial ulcer and prevents recurrence of ulcer at the same site or different site. In our study, 34.6% of subjects did not even use the foot wear at all.

Studies by Moss and colleagues[84] found smokers of younger age were more likely to ulcerate in diabetic patients. In Wisconsin study [84], there was a borderline significance between smoking and ulceration in diabetic patients. In our study, 42% of study subjects were smokers.

In our study 54.3% had arterial insufficiency. This is in accordance with studies by Walters et al [79], Kumar et al [80], Boyko et al and Abbott et al.

Studies by Walters [79], Litzelman , Kumar [80], Carrington , Abbott et al, Boyko [85], Kastenbauer have found neuropathy is a risk factor for amputation in diabetic ulcer. In our study 76.5% of subjects had neuropathy detected by tuning fork test and Biothesiometer.

Studies by Carrington et al [86], show that strict glycaemic control can prevent amputation in diabetic foot. In our study, 55.55% of subjects had a blood glucose level of more than 200mg/dl.

Fernando DJS et al [87], have found diabetic nephropathy is a factor for foot ulceration sometimes progresses to amputation. In our study, 29.6% had diabetic nephropathy.

Serum albumin level more than 3gms% is necessary for adequate wound healing to occur (Dickhaut SC et al [88]). In our study, 83.9% of subjects had a serum albumin level less than 3gms%.

Osteomyelitis can present with the failure of the local part to heal. If there is extensive soft tissue loss, bone may be infected. Usually bone is infected by the way of blood spread. Whereas in diabetic patients , bone involvement occurs

due to infection spread form without inwards ie, from the overlying soft tissue to the deeper bone (Berendt AR[89], Lipsky BA[90] et al). In our study 35.8% of subjects had evidence of osteomyelitic changes on imaging.

Studies by Lipsky BA et al , Borrero E , Goldstein EJ, Viswanathan V [91] et al shows that staphylococcus aureus is the most important pathogen followed by coagulase negative staphylococcus. With optimal microbiological techniques , most of these infections are noted to be polymicrobial. In our study, 32.09% of subjects had polymicrobial infection, and 16.09% of subjects had infection with staphylococcus aureus. However, studies by Cunha BA [92] et al, have found organisms like enterococci, and pseudomonas aeruginosa, are often colonizers rather than pathogen, and antimicrobial therapy specifically targeted against them may not be required.

STEPS TOWARDS PREVENTION

The high prevalence of foot infections and the subsequent economic burden on an already strained economy, gives rise to urgent need to promote foot care in a country like India. The essential step in this goal is early diagnosis of loss protective sensation and ischemia. Surface examination of the foot to diagnose deformities, skin involvement is essential . Toe-nail should be properly taken care of. Injuries caused by toe nails, nail infections and onychocryptosis should be identified.

Life-long care regarding the type of foot wear used should be exercised. The aim of footwear is prevention of ulcer formation, prevent re-ulceration and new site ulcer formation. Nearly 80% of the patients can be benefited from 'over-the-counter' sport shoes with custom-made insoles. Only patients with severe deformities require custom-molded shoes. The biomechanical goal is load reduction and weight transference.

Foot before ulcer formation requires meticulous and scientific care by self and also by the clinician. The management team members include

- Podiatrist
- Internist
- Ophthalmologist
- Endocrinologist

- Infectious disease specialist
- Cardiologist
- Nephrologists
- Vascular surgeon
- Orthopedic surgeon
- Nurse (educator, wound care, and home care)
- Pedorthist/orthotist.

Frequent education of patients and family members is essential. This includes knowledge regarding glycaemic control, diet plans , proper foot wear and need for early identification of early foot lesions. Primary, Secondary and tertiary levels of care are required to be imparted into the patients knowledge so as not to burden the clinician alone with the sole responsibility of care of the patient. Health care personnel education is equally important. A recent study from Chennai reported that of 1259 patients at previous high risk and who presented with foot ulcers, healing occurred in 82% who were judged to have adhered to foot ulcer care advice, compared to 50% of those who did not[73]. Patients should be given ample advice on glycaemic control, smoking reduction, regular exercise, dietary pattern changes, use of low-dose aspirin and statin therapy.

The Himalayan psychosocial and economic costs of Diabetes foot diseases can be brought down by simple preventive measures. Though it may

not be possible to completely prevent ulceration, it is definitely possible to prevent progression of small lesions to the level of amputation.

The KEY elements of preventive care are :

- Yearly examination by health care providers to determine risk factors
- Examination of at-risk feet at each visit
- Patient awareness on daily foot care and diabetes management
- Establishment of specialized clinics to concentrate exclusively on diabetic patients

  60% of patients with ulcer will have recurrence of ulcer in the same region. Despite wide-spread availability of newer therapeutics and diagnostics, the effective prevention of reulceration is paramount in reducing the social and economic burden caused by diabetic foot ulcers.Therapeutic foot wear plays a key role in this context. The Achilles heel in attaining this elusive target still remains the patients lack of knowledge and non-compliance with advice of expert health-care personnel.

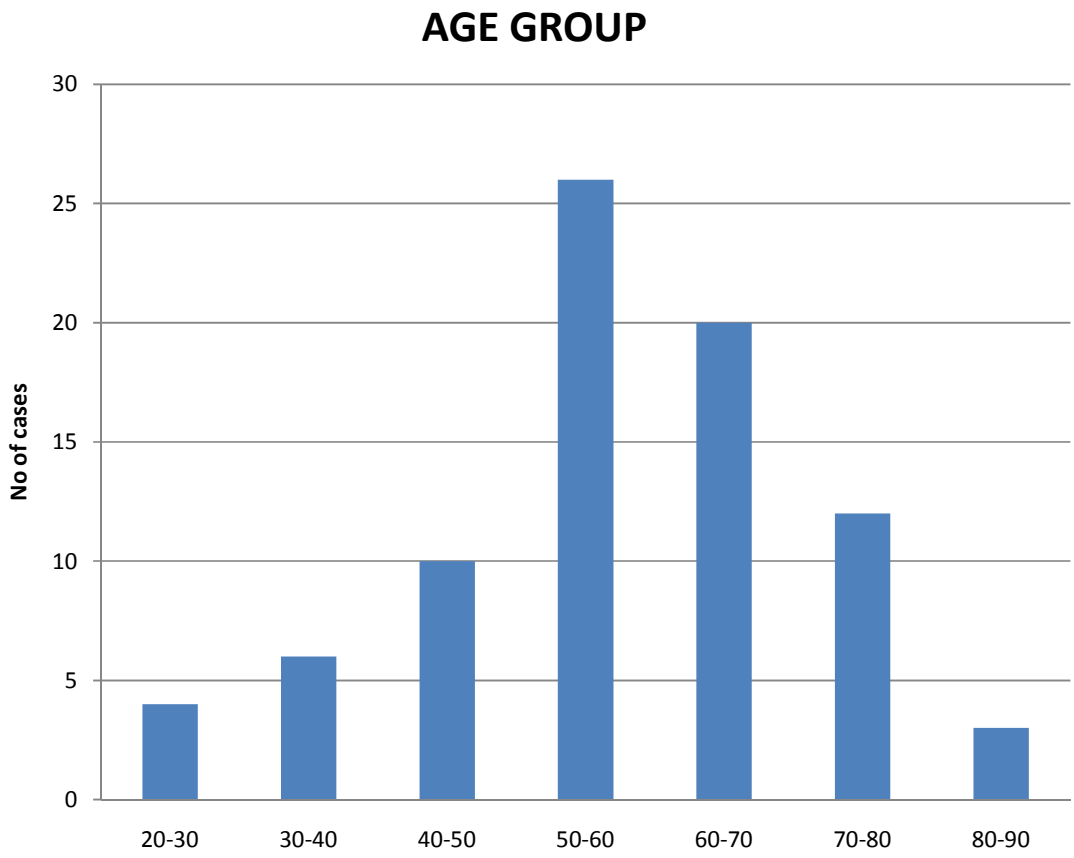
CONCLUSION

- The majority of the amputees in our study were males. The number of the cases being 48 males and 33 females. This workout to a preponderance ratio of 1.46: 1.
- Amputations were more commonly done in the age group of 50 to 60 years. Rehabilitation of these patient subset proves to be a difficult task given the occurrence of numerous other co-morbidities such as hypertension, coronary heart disease and neurological disorders etc.
- There seems to be apparent reduction in the rate of amputation in the older age groups. This observation cannot be projected with certainty because of the fact that these older age group subjects do not present to the hospital in most cases,and also they are less prone to ulceration due to the generalized decrease in the ambulatory capacity.
- Longer the patient with diabetes, the more chance of landing up with amputation. In our study , 45 out of 81 patients underwent major amputation , having diabetes for more than 10 years duration.
- In our study, 36 out of 81 patients had previous history of minor amputations.

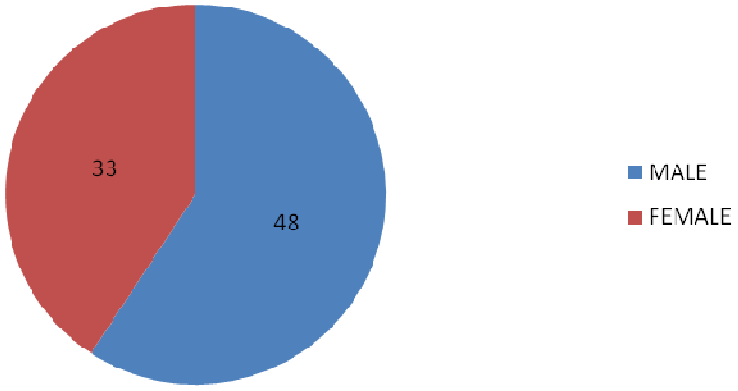
- 65.4% of our study subjects who were infact using footwear, still ended up with ulcerations leading to major amputation. This is mostly due to lack of knowledge regarding proper and adequate foot care.
- A vast majority of our study subjects had peripheral neuropathy (76.5%) as a risk factor for amputation, rather than chronic arterial insufficiency (54.3%).
- 68 out of 81 cases had profound malnutrition, indicated by serum albumin level of less than 3 gms. Improvement of the nutritional status helps to overcome sepsis and promote wound healing.
- Majority of the infections in diabetic ulcers were polymicrobial followed closely by the presence of Pseudomonas aeroginosa.
- Osteomyelitis in a diabetic foot is very difficult to treat and frequently necessitates amputation. Among the study subjects 29(35.8%) had evidence of osteomyelitis on imaging.

Amputation often becomes a palliative or life saving procedure. Major amputations in the younger age group has devastating socioeconomic consequences. Advances in prosthesis and rehabilitation can lessen this burden. Still the most cost effective methods are directed at prevention.

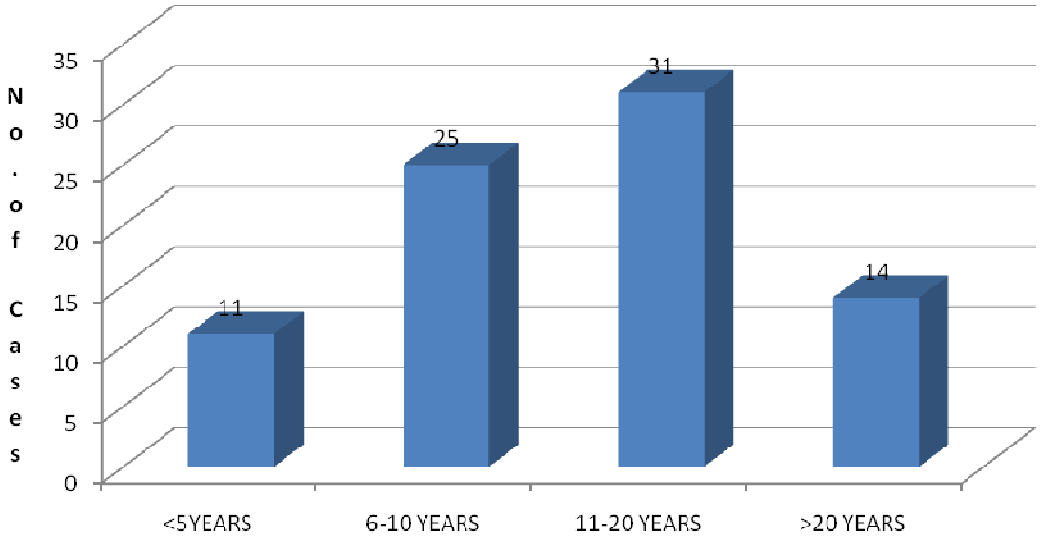
TABLES


AGE GROUP
No of cases
30
25
20
15
10
5
0
20-30
30-40
40-50
50-60
60-70
70-80
80-90

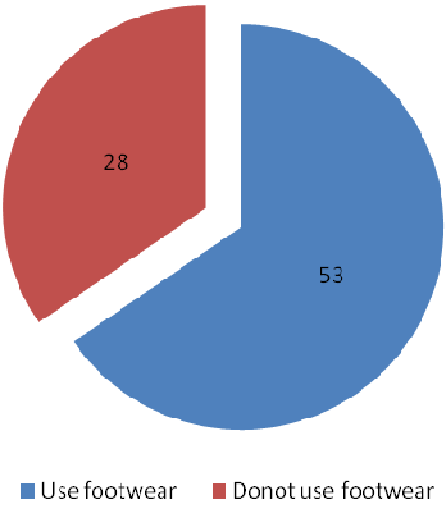
SEX DISTRIBUTION



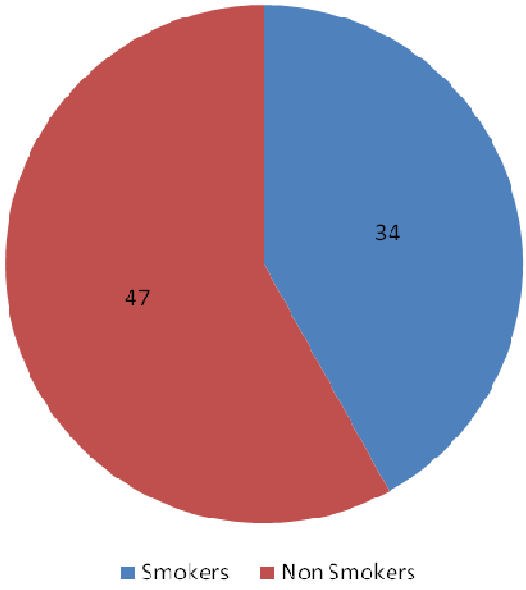
DURATION OF DIABETES

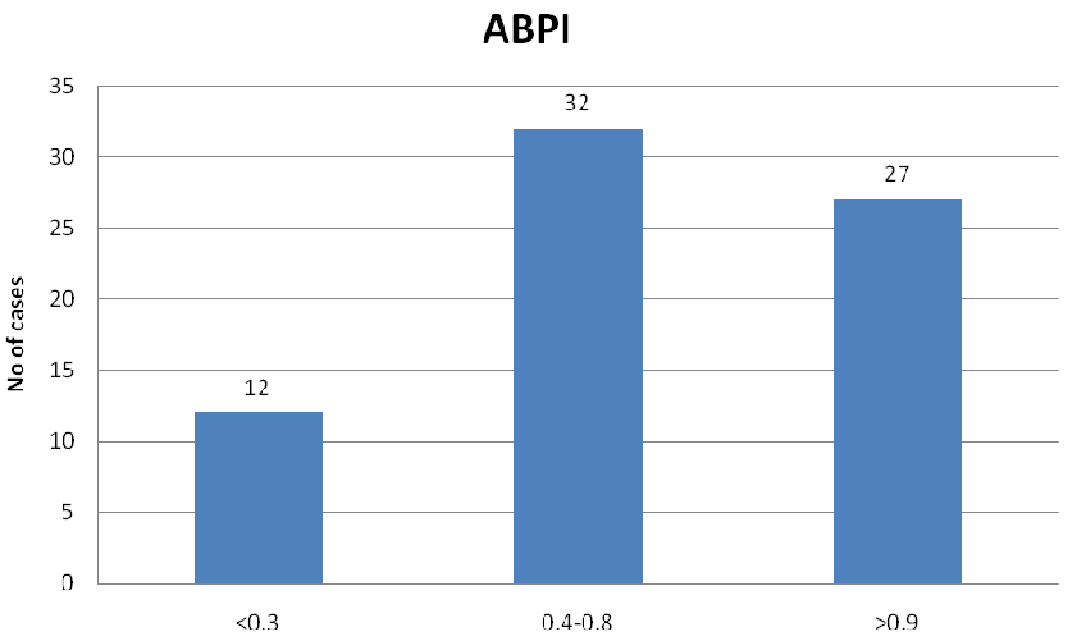


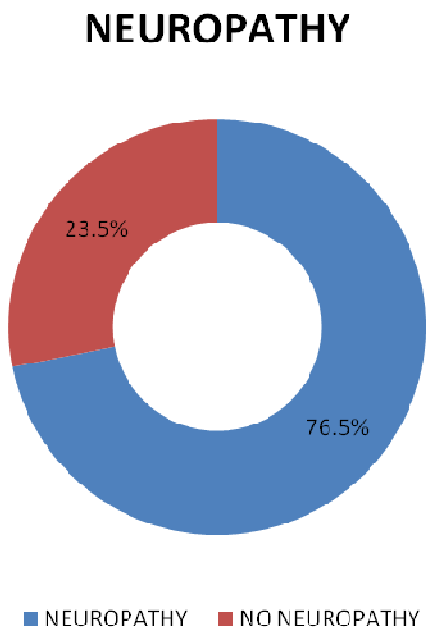
USE OF FOOTWEAR



SMOKING




ABPI
35
30
25
20
15
10
5
0
No of cases
12
32
27
<0.3
0.4-0.8
>0.9


NEUROPATHY
23.5%
76.5%
NEUROPATHY
NO NEUROPATHY

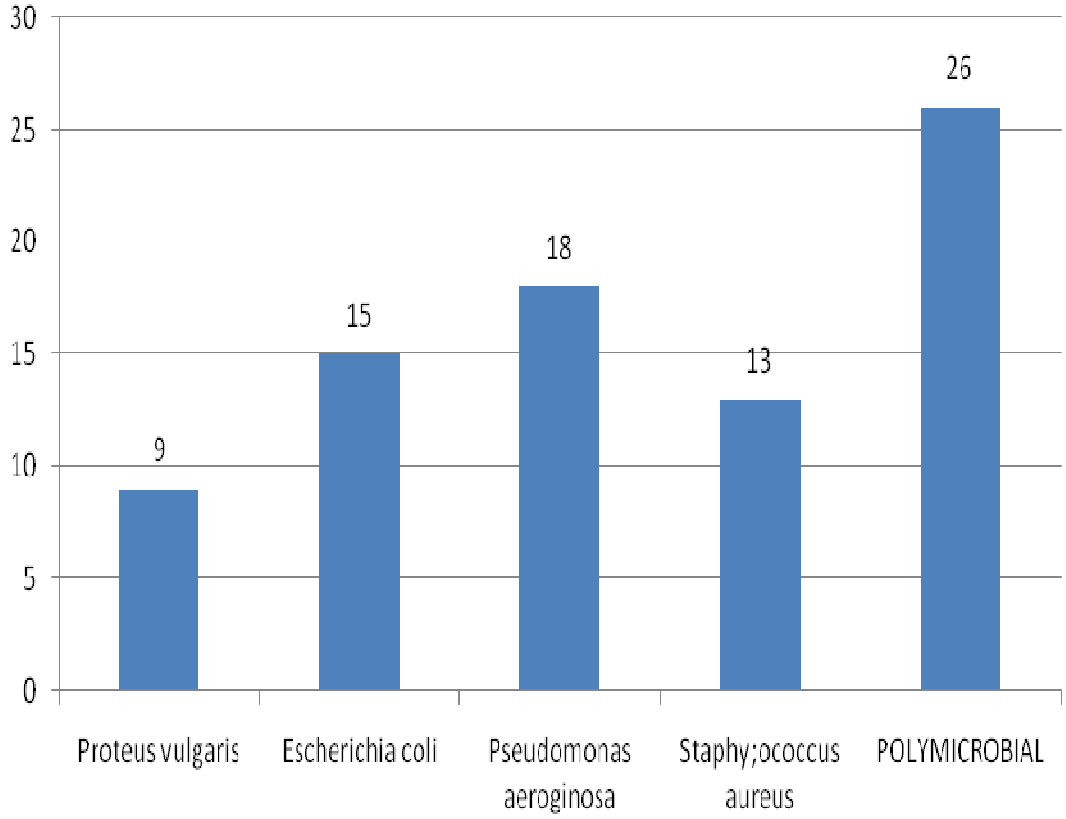

MICROORGANISM
30
25
20
15
10
5
0
9
15
18
13
26
Proteus vulgaris
Escherichia coli
Pseudomonas aeroginosa
Staphy;ococcus aureus
POLYMICROBIAL

IMAGE 1: CALLUS

IMAGE 2 : NAIL DEFORMITY

IMAGE 3: CLAW TOES

IMAGE 4: CORN FOOT

IMAGE 5: HALLUX VALGUS

IMAGE 6 : PRESSURE POINTS

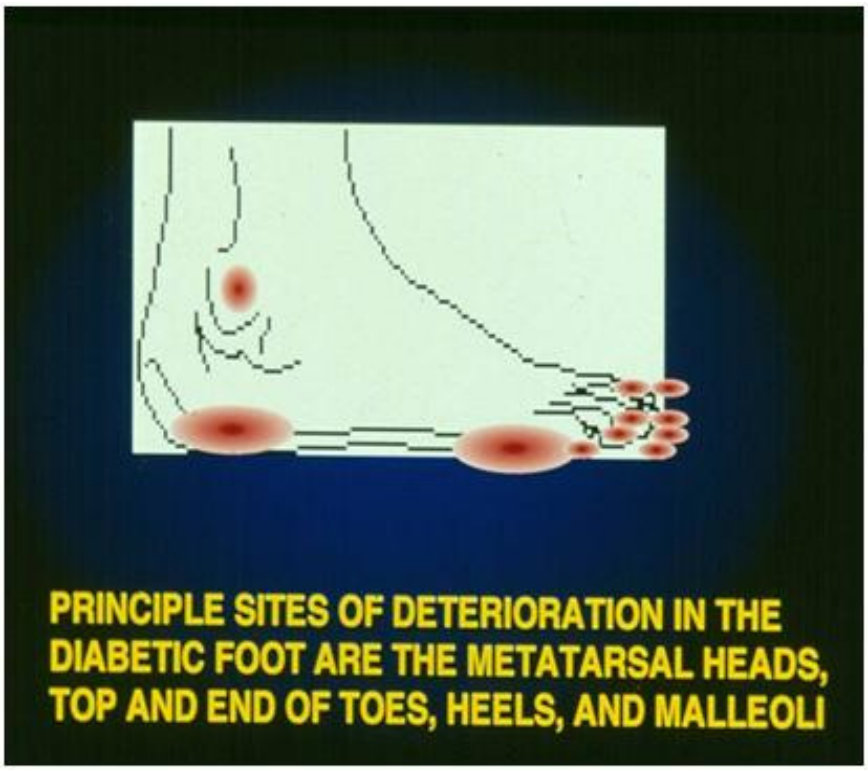
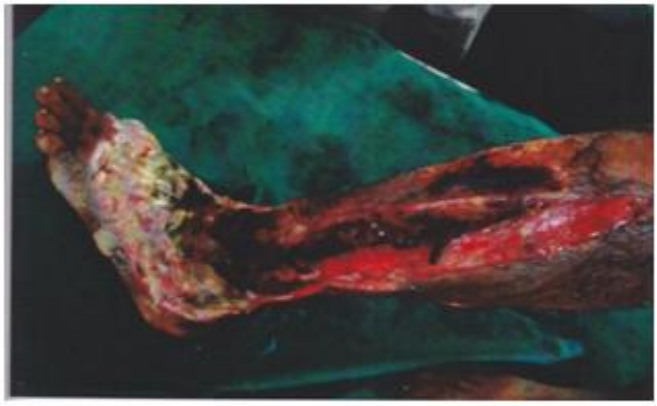


IMAGE 7: CHARCOT FOOT WITH ULCER

IMAGE 8: TOTAL CONTACT CAST

IMAGE 9: EXTENSIVE ULCER



ULCER INVOLVING FOOT & ANKLE

IMAGE 10: MONOFILAMENT TEST

IMAGE 11: BIOTHESIOMETER

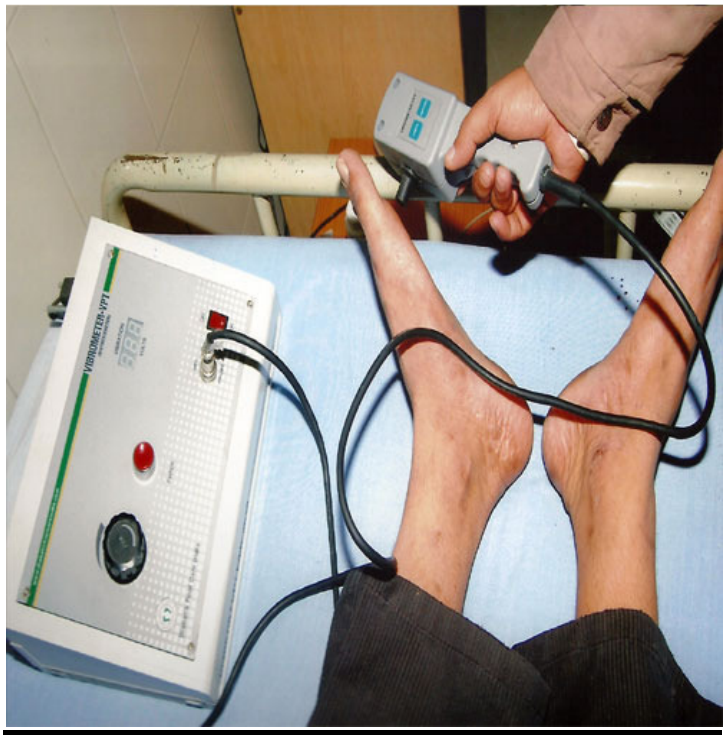


IMAGE 12: NEUROPATHIC ULCER

IMAGE 13: BELOW KNEE STUMP

IMAGE 14: ABOVE KNEE STUMP

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PROFORMA

HISTORY:

1. Age

2. Sex

3. Duration of Diabetes Mellitus

4. Educational Status

5. Smoking Habit

6. Foot Wear Use

7. Previous H/O minor Amputation

CLINICAL EXAMINATION:

1. Peripheral Pulses

2. Site of the ulcer

3. Extent of the Leison

4. Ankle Brachial Pressure Index

5. Neuropathy assessment (Tuning fork / Biothesiometer)

6. Foot deformities

INVESTIGATIONS:

1. Random Blood Sugar

2. Hb%

3. Serum Creatinine

4. Serum Albumin

5. Swabs from all foot lesions for C&S

6. X-ray of the local part.

KEY TO MASTER CHART

History of previous amputation:

- A: Nil
- B: Toe disarticulation
- C: Ray amputation
- D: Mid tarsal amputation
- E : Syme's amputation

Literacy

- A: illiterate
- B: Primary School
- C: Higher Secondary
- D: Degree

Pus culture and sensitivity

- A: Proteus mirabilis
- B: E. coli
- C: Pseudomonas aeroginosa
- D: Staphylococcus aureus
- E: Polymicrobial infection

Foot wear

- Y- Yes
- N- No

Smoking

- Y - Yes
- N - No

Neuropathy

- Y - Yes
- N - No

Osteomyelitis

- Y – Yes
- N - No

MASTER CHART

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R
1																		Pus
2	No.	Name	Age	Sex	IP n0.	Yrs	Prev ampu	Literacy*	Footwear	Smoke	ABPI	Neuropathy	Hb	RBS	Creatinine	S. Alb	Osteomy	C&S
3	1	JEYABALAN	56	M	43910	16	A	B	Y	Y	1	Y	12	132	0.8	2.4	N	B
4	2	NARMATHA	42	F	47194	2	C	B	N	Y	0.7	Y	8	240	1.6	1.6	Y	E
5	3	JEYALAKSHMI	24	F	55178	4	E	D	Y	Y		N	13	138	0.7	2.5	N	C
6	4	MURUGESWARI	61	F	32188	21	A	A	Y	Y	1	Y	9	96	0.9	2.8	Y	E
7	5	GAJENDRAN	57	M	37666	13	C	B	N	Y		Y	14	134	2	2	N	B
8	6	KARUTHAMMAL	66	F	36899	24	E	A	Y	N	1	Y	7	128	0.9	1.6	N	C
9	7	JESUDOSS	59	M	48777	16	C	B	Y	Y	0.8	Y	10.4	121	0.8	2.6	N	E
10	8	KANAGALAKSHMI	56	F	56544	5	D	B	Y	N	1	Y	9	234	1.8	2.6	N	B
11	9	RAMANATHAN	22	M	38769	5	A	D	N	N	0.5	Y	12.4	259	0.7	1.8	N	A
12	10	DEIVANAYAGAM	67	M	52314	5	C	A	Y	Y	1	N	6	89	1.4	2.3	N	E
13	11	PANDI	44	M	47689	4	D	B	Y	N	0.6	Y	11	234	0.9	1.7	Y	C
14	12	RATHINAM	34	F	39936	3	D	C	N	N	1	N	6	320	2.6	2.6	N	A
15	13	MURUGESAN	54	M	41707	13	A	B	Y	Y	0.7	Y	13	180	0.9	1.8	N	E
16	14	HANGAPANDIAMM	64	F	41124	25	B	B	N	N		Y	11.6	240	3	2.4	N	A
17	15	JOSEMIN	25	F	45677	5	A	D	Y	N	0.5	Y	9	79	0.8	2.8	N	B
18	16	KATTURAJA	57	M	41723	14	A	B	Y	Y	0.4	Y	11	214	2.4	2.2	N	C
19	17	BUVANESWARI	33	F	46990	5	C	B	Y	N	0.9	Y	8	245	0.7	1.5	Y	D
20	18	KARUPPASAMY	51	M	53266	11	B	B	N	N	0.7	Y	10.8	83	0.6	1.8	Y	B
21	19	KUTHAPERUMAL	46	M	62788	8	A	B	Y	Y	1	Y	7	234	0.8	2.1	N	D
22	20	CHINNAIAH	68	M	35666	22	D	A	Y	Y	0.8	Y	12.8	256	2.6	1.9	N	C
23	21	NELAMEGUM	58	M	53838	13	B	B	N	Y	1	Y	9	115	0.9	1.6	Y	A
24	22	SUBBULAKSHMI	45	F	53255	8	A	B	Y	N	0.9	Y	11.4	235	3.2	2.2	N	C
25	23	MARY	36	F	60113	4	C	C	N	N		Y	12.2	154	0.9	2.3	N	B
26	24	ADAIKAN	56	M	61866	13	B	B	Y	Y	0.7	Y	8	94	0.9	2.6	Y	D
27	25	SOUNDRAPANDI	64	M	70829	26	A	B	N	N	1	Y	7	244	0.8	1.8	N	E
28	26	THASNEEM	56	F	65790	11	B	B	Y	N	0.9	Y	7	86	1	2.2	N	C
29	27	SUMATHI	43	F	72761	6	A	C	Y	N	1	N	11	208	0.8	1.7	Y	E
30	28	SUBRAMANIAN	64	M	45688	16	B	B	Y	Y	0.8	Y	6	78	0.8	2.6	N	B

MASTER CHART

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R
31	29	KAMAL MUSTAFAH	57	M	70825	17	B	B	Y	Y	1	Y	12.4	289	1.8	3.2	Y	E
32	30	PANDIAMMAL	68	F	67899	9	A	B	Y	N	0.5	N	7	267	0.7	3.3	N	A
33	31	NAGAPPAN	67	M	27287	16	B	B	N	Y	0.7	Y	7	74	0.9	2.7	N	E
34	32	MUNIYAMMAL	61	F	32284	15	A	B	N	N	0.5	Y	12.8	119	0.9	1.5	Y	B
35	33	VERAN	39	M	37375	7	A	C	Y	N	0.9	N	8	224	1	3.2	N	E
36	34	ARPUTHAM	56	M	36845	9	B	B	N	Y	0.6	N	13	168	0.8	2.7	N	C
37	35	NATARAJ	48	M	43454	8	A	C	Y	Y		Y	7	280	1.4	3.1	N	E
38	36	KARUPPAYEE	67	F	35467	5	B	B	N	N	0.9	N	10	267	0.7	2.6	Y	D
39	37	MUNIARAJ	71	M	66789	25	A	A	Y	N	0.8	Y	13	254	1	1.8	Y	E
40	38	BHAGAVATHY	54	M	56781	6	A	B	Y	Y	0.6	N	9	174	0.9	2.4	N	C
41	39	RAMANATHAN	67	M	43910	13	A	B	Y	Y	0.6	Y	10.6	278	1	2.8	Y	E
42	40	GANAGALAKSHMI	44	F	59917	8	A	B	Y	N	0.9	N	8	84	0.8	2.4	N	B
43	41	NAGAMMAL	57	F	61609	17	B	B	N	N	0.8	Y	11.4	288	1.6	3.2	N	E
44	42	KANAGARAJ	59	M	63697	15	A	B	Y	N		Y	7	256	0.9	2.4	Y	C
45	43	RAJAMEGAM	68	M	73561	14	B	B	N	Y	1	Y	9	180	1.8	1.9	Y	E
46	44	UMA	49	F	76369	7	A	C	N	N	0.4	Y	11	280	1	2.3	N	A
47	45	MAJID SULTAN	36	M	36857	13	C	D	Y	Y	0.8	Y	10.4	118	1.4	3.1	Y	E
48	46	SUJATHA	29	F	55609	7	A	D	N	N	0.2	N	7	280	0.9	2.5	N	C
49	47	THIRUMALAI	58	M	89076	8	B	B	Y	N	0.5	N	9	240	0.9	1.6	Y	E
50	48	JOHN	58	M	56789	8	B	B	N	Y	0.3	Y	10.4	260	0.8	2.9	N	D
51	49	GOMATHI	49	F	45543	6	A	B	Y	N	0.6	N	8	124	1.6	3.2	N	B
52	50	SUNDARAM	54	M	15560	13	B	B	Y	Y		Y	11.4	280	0.7	2.8	Y	E
53	51	SAROJA	54	F	35609	8	A	C	Y	N	0.7	N	7	250	0.8	3.1	N	A
54	52	RAMAR	69	M	10922	16	A	B	N	Y	0.3	Y	11.4	240	0.9	2.6	N	E
55	53	AROCIAMARY	65	F	16534	8	B	B	Y	N	0.9	Y	11	180	0.8	1.8	Y	D
56	54	NAGAPPAN	48	M	16476	8	A	B	N	Y	0.6	N	10.2	226	2.2	2.5	N	A
57	55	SUBRAMANIAN	56	M	16494	15	A	C	Y	N	0.2	Y	9	240	0.7	3.2	Y	E
58	56	RATHNAVEL	74	M	19480	14	A	A	Y	N	0.3	Y	9	229	2.2	2.7	N	B
59	57	PANDIAN	68	M	29875	16	B	B	Y	Y	0.8	Y	8	114	0.8	3.3	Y	E
60	58	CHITRA	31	F	29887	9	A	D	Y	N	0.9	Y	10	236	2.4	2.4	Y	D

MASTER CHART

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R
61	59	KALEEL IBRAHIM	75	M	46578	23	B	A	N	Y	0.3	Y	11.8	108	0.9	1.9	N	E
62	60	KASTURI	59	F	68795	7	A	C	N	N	0.9	N	7	240	1.8	2.3	N	B
63	61	JEYABALAN	69	M	45612	8	A	B	Y	Y		Y	11	260	1	1.6	Y	C
64	62	POTHUMPONNU	79	F	32789	25	A	A	N	N	0.9	Y	7	250	0.8	2	N	D
65	63	MUNUSAMY	69	M	25879	6	C	B	Y	Y	0.6	Y	10.8	290	2.4	2.6	Y	B
66	64	RAMAMOORTHY	61	M	43143	7	B	B	Y	N	0.1	Y	7	120	0.9	3.2	N	D
67	65	VEDAMMAL	57	F	51889	8	A	C	Y	N		N	10.8	260	1.1	2.8	N	C
68	66	KARUPANAN	77	M	77954	26	A	A	Y	Y	0.7	Y	14	233	1	2.8	N	B
69	67	PANCHU	73	F	78017	29	A	A	Y	N	0.9	Y	11.4	246	1	1.8	Y	E
70	68	PRABHU	71	M	53458	28	A	B	Y	Y	0.2	Y	9	272	0.9	2.6	Y	C
71	69	SUNDARAJAN	56	M	42365	15	A	C	N	N	0.7	Y	7	128	0.9	2.1	N	D
72	70	GANESAN	76	M	15759	14	A	B	Y	N	0.9	Y	13	116	0.9	1.9	N	E
73	71	MANIKANDAN	86	M	65241	25	B	A	N	Y	0.3	Y	12	132	0.7	3.4	N	C
74	72	PANDIAMMAL	75	F	28030	29	A	A	Y	N	1	N	8	136	1.5	2.8	Y	B
75	73	ASHOKAN	83	M	34150	15	B	A	Y	N	0.6	Y	6	88	0.9	1.6	N	A
76	74	LAKSHMI	55	F	43100	8	A	C	N	N	0.2	Y	12.4	124	0.6	2.6	N	D
77	75	RUKMANI	73	F	43663	30	B	A	Y	N		Y	10.4	114	1	3.2	Y	E
78	76	SEKAR	53	M	44953	9	A	C	Y	Y	0.7	N	8	160	0.7	2.4	N	C
79	77	JEYALAKSHMI	83	F	50284	16	A	B	Y	N	0.3	Y	12.8	289	0.8	1.8	N	D
80	78	SRINIVASAN	57	M	28097	14	B	B	N	N	0.7	Y	8	240	1.6	2.6	N	C
81	79	SIVAKUMAR	71	M	79394	16	A	B	Y	N		Y	7	78	0.9	1.9	N	E
82	80	PANDI	78	M	82527	15	B	B	Y	N	0.6	Y	11	220	0.8	3.1	Y	C
83	81	RAJEENABEGUM	56	F	73427	11	D	B	N	N	0.9	Y	10.2	240	2	2.1	N	D